

Molecular Biology

THE ROLE OF *algZ* IN THE REGULATION OF *PSEUDOMONAS AERUGINOSA* TWITCHING MOTILITY

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Pseudomonas aeruginosa is an opportunistic pathogen of both burn patients and individuals with the genetic disease cystic fibrosis. Virulence factors shown to be important in the disease process include the production of the exopolysaccharide alginate and twitching motility (TM) or flagellar-independent movement across solid surfaces. AlgZ, a *P. aeruginosa* regulatory protein, binds sequences upstream of the alginate biosynthetic operon and activates transcription of these genes. Recently, *algZ* has been found to be essential for TM, which utilizes type-4 pili.

This study investigates the mechanism by which *algZ* regulates TM. AlgZ may either activate a gene(s) that is required for TM or repress a gene(s) that negatively controls TM, or both. To determine how AlgZ regulates TM, a genetic approach was used. A transposable promoter probe containing a promoterless *lacZ* gene and a gentamicin resistance marker was utilized. The transposon (Tn) was introduced via mating into a *P. aeruginosa* strain containing an arabinose-inducible *algZ*. Gentamicin resistant bacteria were then examined to determine if a gene(s) regulated by *algZ* had been interrupted. These colonies were grown in the presence and absence of arabinose on media containing X-gal. Genes negatively regulated by *algZ* should appear blue in the absence of arabinose and white in the presence of arabinose since the presence of AlgZ would lead to the repression of *lacZ*. Alternatively, if the interrupted gene is positively regulated by *algZ*, the colony will appear blue in the presence of arabinose and white in the absence of arabinose. Of 94 gentamicin resistant colonies examined, 36 were white in the presence of arabinose and blue in its absence. These preliminary data suggest that AlgZ represses a repressor gene(s) involved in TM. These colonies are being further examined to determine their TM phenotype and the identity of the interrupted gene(s).